

C H A P T E R 5

Theories and Applications of Pavlovian Conditioning

HE NEVER SAW IT COMING

Clarence started dating Felicia several months ago. Their very different personalities—Clarence is quiet and shy, and Felicia is outgoing and social—did not keep them from becoming close. Felicia introduced Clarence to a social life he had never before experienced; they enjoyed romantic dinners and exciting parties. Felicia was an excellent dancer, which made dancing fun. Clarence was not used to partying late. He had to get up early for work and felt drained the entire day after a late night out. Yet the late nights did not seem to bother Felicia, and she was wide awake the next day, even with only a few hours of sleep. Last month, Felicia started to drink a lot of alcohol. Clarence rarely drinks more than a beer or two, and he was quite concerned about Felicia's drinking. She called him a lightweight for only having one or two drinks. Clarence did not mind the teasing, but he really did not want to drink much. He thought he was in love with Felicia; however, he was troubled by some of her actions toward him. The last several times they were out, Felicia became angry and verbally abusive to him for no apparent reason. The verbal comments were very harsh, and Clarence was hurt. The next day, Felicia apologized for her behavior and promised not to be nasty again. Yet the next time that they went out, Felicia again became angry and abusive. Clarence could not figure out why she became hostile, and he considered breaking off their relationship. He mentioned his concerns to his friend Jared. To Clarence's surprise, Jared said he knew why Felicia became hostile: The alcohol was to blame. Jared also said he felt frightened when Felicia started to drink. Clarence was not sure that Jared was right, but he decided he would pay close attention to the amount Felicia drank and whether she became hostile afterwards. He was hopeful that this was the reason for her hostility, but he was not sure what he would do if it was.

Why did Clarence fail to see the relationship between Felicia's drinking and her hostility? The answer may lie in a phenomenon called the CS preexposure effect. When a stimulus is first



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presented without a UCS, subsequent conditioning is impaired when that stimulus is presented with the UCS. Clarence had seen people drinking without becoming aggressive, and this experience may have caused him to fail to recognize the relationship between Felicia's drinking and her hostility. We examine the CS preexposure effect later in the chapter; our discussion may tell us what caused Clarence to fail to associate the sight of Felicia's drinking (CS) with her hostility toward him (UCS).

Clarence's friend Jared recognized the relationship between Felicia's drinking and her hostility. As a result of this recognition, Jared became frightened when he saw Felicia drinking. One of the main questions we will address in this chapter is why Jared was able to associate Felicia's drinking and her hostility. We will answer this question by examining the nature of Pavlovian conditioning.

THEORIES OF PAVLOVIAN CONDITIONING

Pavlov (1927) conducted an extensive investigation of the principles governing the acquisition and extinction of a conditioned response. During the past 30 years, many studies have examined both how conditioned responses are acquired and whether the CR is similar or different from the UCR. This research has challenged Pavlov's assumptions regarding both the nature of conditioning and the conditioned response. New theories have emerged to explain these recent research findings.

The Nature of the Conditioned Response

One important question in Pavlovian conditioning concerns the nature of the conditioned response. Is the CR just the UCR elicited by the CS? Or is the CR a behavior distinctively different from the UCR?

Stimulus-Substitution Theory

Pavlov (1927) suggested that as a result of conditioning, the conditioned stimulus becomes able to elicit the same response as the unconditioned stimulus. Why would Pavlov assume that the CR and UCR were the same response? Pavlov was observing the same digestive responses (e.g., saliva, gastric juices, insulin) as both the CR and UCR. The fact that both the CS and UCS elicit similar responses logically leads to the conclusion that the CR and UCR are the same.

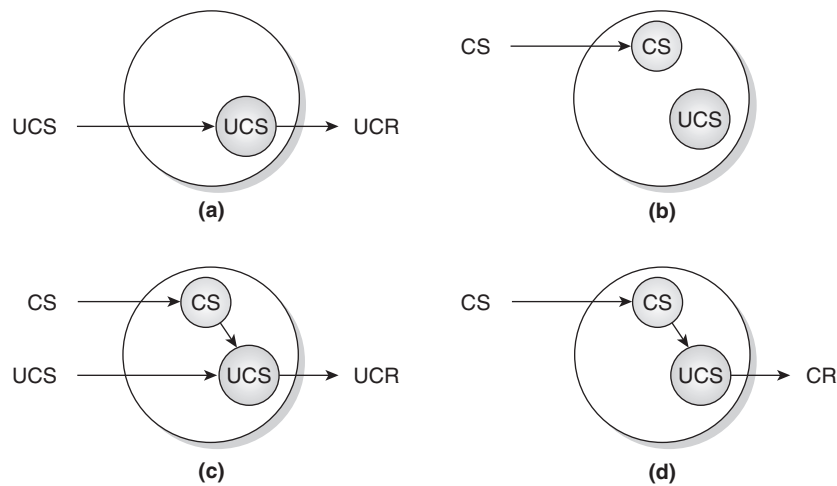
How does the CS become able to elicit the same response as the UCS? According to Pavlov (1927), the presentation of the UCS activates one area of the brain. Stimulation of the neural area responsible for processing the UCS leads to the activation of a brain center responsible for generating the UCR. In Pavlov's view, an innate, direct connection exists between the UCS brain center and the brain center controlling the UCR; this neural connection allows the UCS to elicit the UCR.

How might the connection between the CS and CR develop? When the conditioned stimulus is presented, it excites a distinct brain area. When the UCS follows the CS, the brain centers responsible for processing the CS and UCS are active at the same time. According to

Pavlov (1927), the simultaneous activity in two neural centers leads to a new functional neural pathway between the active centers. The establishment of this neural connection causes the CS to activate the neural center processing the CS, which then arouses the UCS neural center. Activity in the UCS center leads to activation in the response center for the UCR, which then allows the CS to elicit the CR. In other words, Pavlov is suggesting that the CS becomes a substitute for the UCS and elicits the same response as the UCS; that is, the CR is the UCR, only elicited by the CS instead of the UCS. Figure 5.1 provides an illustration of Pavlov's **stimulus-substitution theory** of conditioning.

Pavlov's stimulus-substitution theory proposes that the CS elicits the CR by way of the UCS. Holland and Rescorla's (1975) study provides strong support for this view. In their study, two groups of food-deprived rats received tone CS and food UCS pairings. After conditioning, one group of rats was fed until satiated, while the other group remained food deprived. The animals then received a series of CS-alone extinction trials. Holland and Rescorla reported that the CS elicited a weaker CR in the satiated than in the hungry rats. Why did the removal of food deprivation reduce the strength of the CR? According to Holland and Rescorla, food satiation reduces the value of food and thereby reduces the ability of the UCS to elicit the UCR. The reduced value of the UCS causes the CS to elicit a weaker CR.

FIGURE 5.1 Pavlov's stimulus-substitution theory of classical conditioning. (a) The UCS activates the UCS brain center, which elicits the UCR; (b) the CS arouses the area of the brain responsible for processing it; (c) a connection develops between the CS and UCS brain centers with contiguous presentation of CS and UCS; and (d) the CS elicits the CR as a result of its ability to activate the UCS brain center



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The Conditioning of an Opponent Response

While the conditioned and unconditioned responses are often similar, in many cases, they seem dissimilar. For example, the conditioned response of fear differs in many ways from the unconditioned response of pain. While both involve internal arousal, the sensory aspects of the two responses are not the same. Warner's 1932 statement that "whatever response is grafted onto the CS, it is not snipped from the UCS" indicates a recognition of CR and UCR differences.

The research of Shepard Siegel and his colleagues represents the most impressive accumulation of evidence suggesting that the conditioned and unconditioned responses are different (Siegel, 1978, 1991, 2001; Siegel, Baptista, Kim, McDonald, & Weiss-Kelly, 2000; Siegel, Hinson, & Krank, 1978; Siegel & Ramos, 2002). In several of their studies, Siegel and his associates used morphine as the unconditioned stimulus (Siegel, 1978; Siegel et al., 1978). **Analgesia**, or reduced sensitivity to pain, is one unconditioned response to morphine. Siegel reported that the conditioned response to stimuli, such as lights or tones, that have been paired with morphine is **hyperalgesia**, or an increased sensitivity to pain.

How did Siegel know that a conditioned stimulus associated with morphine makes an event more unpleasant? To illustrate both the analgesic effect of morphine and the hyperalgesic effect of a stimulus paired with morphine, Siegel placed a rat's paw on a hot plate and measured how long it took the rat to remove its paw. He observed that rats injected with morphine (the UCS) took longer to remove their paws from the heated plate than did animals that had not received the morphine injection. The light or tone paired with morphine, by contrast, caused the rats to remove their paws more quickly than did animals that had been presented with a stimulus not paired with the morphine (UCS).

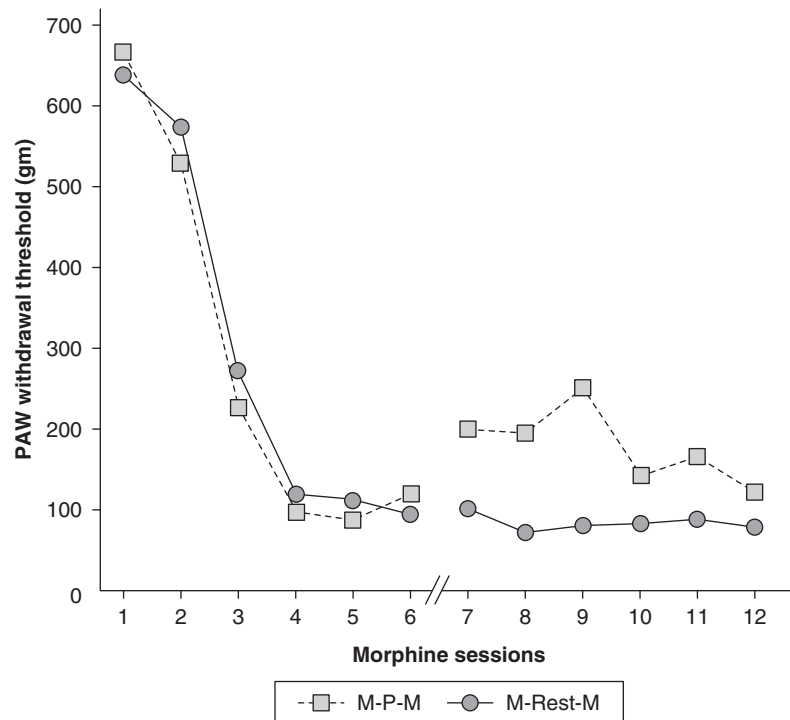
Siegel (1975) also found that while the UCR to insulin is hypoglycemia, the CR to a stimulus paired with insulin is hyperglycemia. Additional studies reported that the UCR to alcohol is hypothermia, while the CR to a stimulus associated with alcohol is hyperthermia (Crowell, Hinson, & Siegel, 1981; Le, Poulos, & Cappell, 1979).

This research suggests not only that the CR can be the opposite of the UCR, but also that conditioning is responsible, at least in part, for the phenomenon of **drug tolerance** (Siegel, 2001; Siegel et al., 2000). Tolerance to a drug develops when, with repeated use of a drug, the effectiveness of the drug declines and larger doses are necessary to achieve the same pharmacological effect (see Chapter 3). According to Siegel, tolerance represents the conditioning of a response that is opposite to the unconditioned drug effects. Thus, the environmental cues present during drug administration antagonize the drug's action and result in a lower pharmacological reaction to the drug. The interoceptive cues that occur early in the drug experience also can become able to elicit a conditioned hyperalgesia response (Sokolowska, Siegel, & Kim, 2002). According to Sokolowska et al., the association of these interoceptive early-onset cues plays an important role in the development of tolerance to morphine.

Two lines of evidence support the idea that conditioning plays a role in drug tolerance. First, Siegel (1977) found that exposure to the CS (environment) without the UCS (drug), once the association has been conditioned, results in the extinction of the opponent CR; the elimination of the response to the CS produces a stronger reaction to the drug itself (see Figure 5.2). Second, Siegel et al. (1978) reported that an increased response to the drug can also be induced by changing the stimulus context in which the drug is administered. The

novel environment does not elicit a CR opposite to the drug's unconditioned effect; in turn, the absence of the opposing CR results in a stronger unconditioned drug effect. A change in context also has been reported to lead to a reduced tolerance to alcohol (Larson & Siegel, 1998) and caffeine (Siegel, Kim, & Sokolowska, 2003). And a reduced tolerance can lead to a heightened drug response. Siegel (1984) reported that 7 out of 10 victims of a drug overdose recalled that a change in environment was associated with the drug overdose, while Siegel, Hinson, Krank, and McCully (1982) observed that a drug overdose typically occurs when an addict takes his or her usual drug dose, but in an unfamiliar environment. Without the protective conditioned opposing response, the effect of the drug is increased, resulting in the overdose (Siegel & Ramos, 2002).

FIGURE 5.2 Tolerance develops to morphine injection (as indicated by lowered pain threshold) during first 6 injection sessions. The presentation of 12 placebo sessions (injections without morphine) in M-P-M (morphine-placebo-morphine) group animals extinguished the conditioned opponent response and reduced tolerance (as indicated by an increased pain threshold). Animals given a 12-day rest period between morphine injections (M-Rest-M group) showed no change in pain threshold from the sixth to the seventh session



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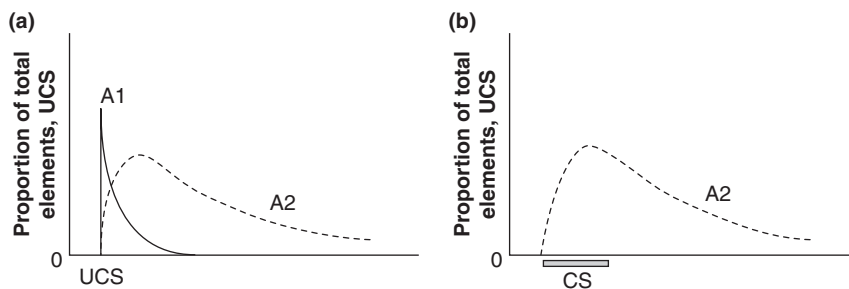
Why is the CR sometimes similar and sometimes dissimilar to the UCR? Allan Wagner's **sometimes-opponent-process (SOP) theory** provides one answer to this question; we will look at his view next.

Sometimes-Opponent-Process (SOP) Theory

Recall our discussion of Solomon and Corbit's (1974) opponent-process theory in Chapter 3. We learned that an event elicits not only a primary affective response, but also a secondary opponent affective reaction. Wagner's SOP theory (Brandon & Wagner, 1991; Brandon, Vogel, & Wagner, 2002; Wagner & Brandon, 1989, 2001) is an extension of opponent-process theory that can explain why the CR sometimes seems the same as and sometimes different from the UCR. According to Wagner, the UCS elicits two unconditioned responses—a primary A1 component and a secondary A2 component. The primary A1 component is elicited rapidly by the UCS and decays quickly after the UCS ends. In contrast, both the onset and decay of the secondary A2 component are very gradual.

The Importance of the Nature of the A2 Response. The secondary A2 component of the UCR can be the same as the A1 component, or the A1 and A2 components can differ. Whether A1 and A2 are the same or different is important. A key aspect of Wagner's view is that conditioning only occurs to the secondary A2 component; that is, the CR is always the secondary A2 reaction (see Figure 5.3). The CR and UCR will appear to be the same when the A1 and A2 components are the same. Different A1 and A2 components will yield a CR and UCR that look different; however, the CR and UCR are really the same in this case. This is true because the A1 component is the response we associate with the UCR. When the A2 reaction is opponent to the A1, it looks as if the CR (A2) and UCR (A1 and A2) are different. Yet, the CR is merely the secondary A2 component of the UCR. Perhaps several examples would clarify this aspect of SOP theory.

FIGURE 5.3 Wagner's SOP theory. (a) The UCS elicits the A1 and A2 components of the UCR. (b) The pairing of the CS and UCS leads to the CS being able to elicit the A2 component of the UCR

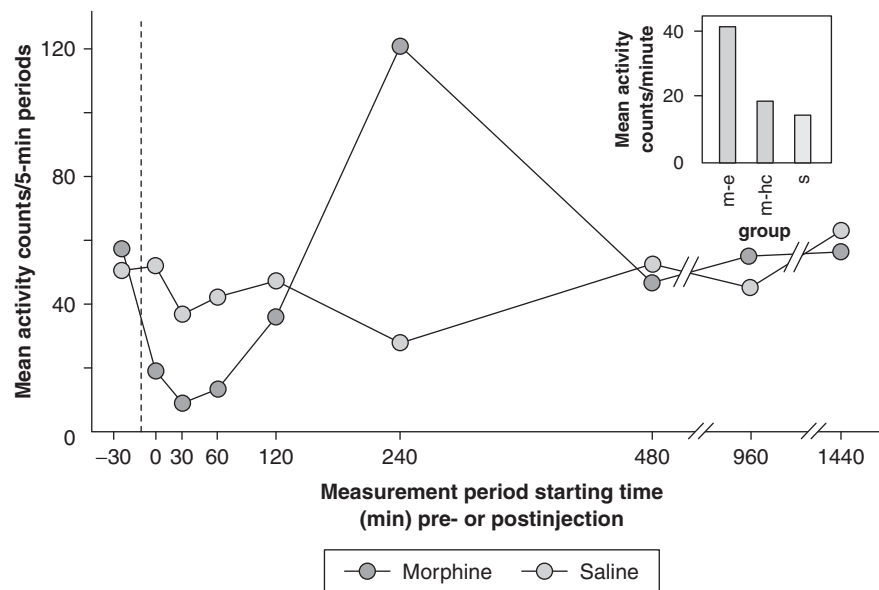


Suppose an animal receives a brief electric shock. The initial reaction to shock is agitated hyperactivity. This initial increased reactivity is followed by a long-lasting hypoactivity or “freezing” response (Blanchard & Blanchard, 1969; Bolles & Riley, 1973). The freezing response, or **conditioned emotional reaction**, is the response conditioned to a stimulus paired with electric shock.

Paletta and Wagner (1986) demonstrated the two-phase reaction of an animal to a morphine injection. The initial A1 reaction to morphine is sedation or hypoactivity. Figure 5.4 shows that the initial activity level is lower in rats given morphine rather than saline. However, two hours after the injection, the morphine-receiving rats are significantly more active than the control rats who received saline.

What is the conditioned reaction to an environmental stimulus paired with morphine? As Figure 5.4 shows, the morphine animals were hyperactive when tested in the environment where they received morphine. Testing the morphine animals in their home cages produced a level of activity comparable to that of control animals not receiving morphine injections. These observations indicate that the conditioned reaction morphine produces is hyperactivity, which is the A2 secondary component of the UCR.

FIGURE 5.4 Illustration of activity levels following injections of morphine or saline. Activity first decreases, then increases above normal after morphine injections. Animals given morphine in a distinctive environment show increased activity, or a conditioned A2 response, when placed in that environment without morphine (shown in bar graph)



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We have looked at two examples in which the A1 and A2 components of the UCR were opposite. In other cases, A1 and A2 are the same. Grau (1987) observed that the unconditioned response to radiant heat consisted of an initial short-duration **hypoalgesia**, or decreased sensitivity to pain, followed by a more persistent hypoalgesia. How do we know that both A1 and A2 reactions to a painful stimulus such as radiant heat are hypoalgesia? The use of the opiate antagonist naloxone can demonstrate this similarity of A1 and A2 response. Naloxone blocks the long-term, persistent hypoalgesia (A2) but has no effect on the short-term, immediate hypoalgesia (A1). This differential effect means that the A1 hypoalgesic response is nonopioid, while the A2 hypoalgesia involved the opioid system. Furthermore, Fanselow and his colleagues (Fanselow & Baackes, 1982; Fanselow & Bolles, 1979) showed that it is the A2 opioid hypoalgesia reaction that is conditioned to environmental stimuli paired with a painful unconditioned stimulus such as radiant heat. These researchers observed that administration of naloxone prior to conditioning prevented the conditioning of the hypoalgesic response to environmental cues that were paired with a painful event.

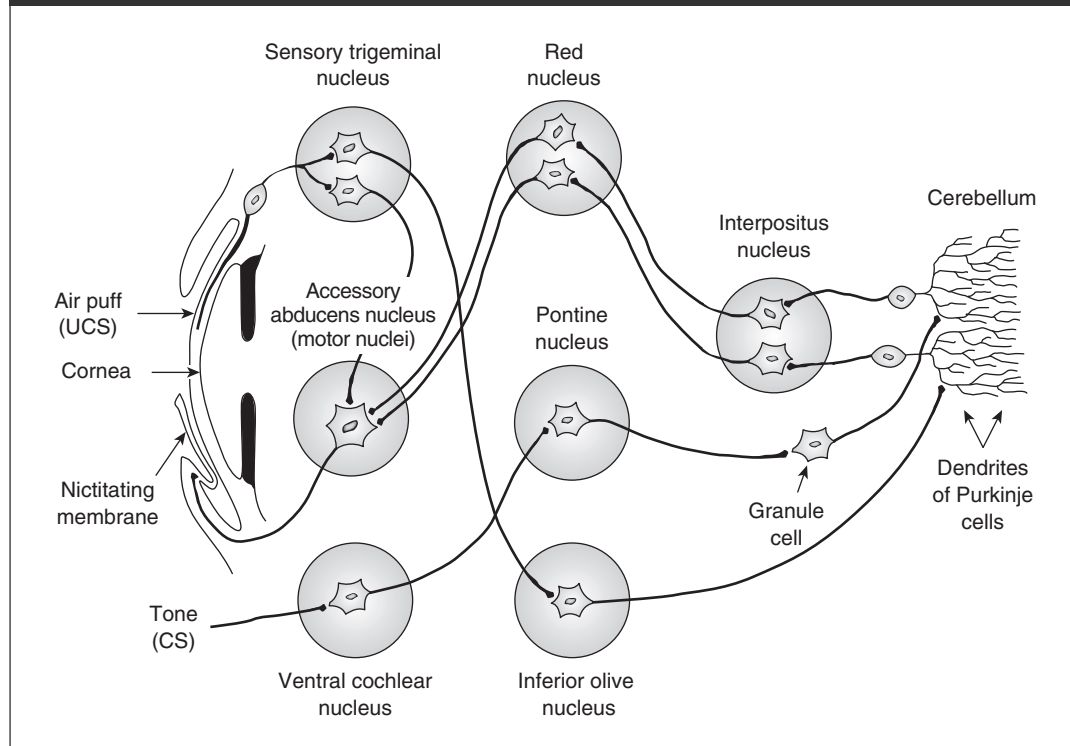
A study by Thompson et al. (1984) provides perhaps the most impressive support for SOP theory. These researchers investigated the conditioning of an eyeblink response to a tone paired with a corneal air puff to a rabbit's eye. They found that two neural circuits mediate the rabbits' unconditioned eyeblink response (see Figure 5.5). A fast-acting A1 response is controlled by a relatively direct path from the area of UCS application on the fifth sensory nucleus to the sixth and seventh motor nuclei controlling the eyeblink response. Stimulation of this neural circuit produces a fast-acting and rapid-decay eyeblink response. A secondary A2 circuit begins at the fifth nucleus and goes through the inferior olive nucleus, several cerebellar structures, and red nucleus before reaching the motor nuclei. Activation of this A2 circuit produces a slow-acting eyeblink response. Thompson and his colleagues also found that destruction of the indirect pathway eliminated a previously conditioned eyeblink response but did not affect the short-latency, unconditioned eyeblink response. Destruction of the indirect A2 pathways also precluded any reconditioning of the eyeblink response.

Backward Conditioning of an Excitatory CR. We learned in Chapter 4 that a forward conditioning paradigm produces a more reliable acquisition of the CR than a backward conditioning paradigm. While this statement is generally correct, Wagner's SOP theory indicates that backward conditioning can yield an excitatory CR if the CS is presented just prior to the peak of the A2 unconditioned response.

Larew (1986) provided strong support for this aspect of Wagner's SOP theory. In Larew's study, rats received a 2-second footshock UCS followed by a 30-second tone. The tone occurred 1 second, 31 seconds, or 60 seconds after the UCS. Control rats received no UCS-CS pairings. Larew observed an excitatory conditioned response with the 31-second UCS-CS backward conditioning procedure, but no excitatory conditioning with either a 1-second UCS-CS interval or a 60-second UCS-CS interval. These results suggest that excitatory conditioning occurs with a backward procedure when the CS immediately precedes the A2 response.

Problems With SOP Theory. Wagner and Brandon (Brandon & Wagner, 1991; Brandon et al., 2002; Wagner & Brandon, 1989, 2001) commented that despite the strong support for SOP theory, some research seems to be inconsistent with this view. One significant problem

FIGURE 5.5 The two neural circuits that mediate the influence of tone (CS) and corneal air puff (UCS) on the nictitating membrane response. The UCS activates a direct route between sensory (trigeminal nucleus) and motor (accessory abducens nucleus) neurons and an indirect route through the inferior olive nucleus, cerebellum (interpositus and dendrites of Purkinje cell), and red nucleus before reaching the motor nuclei controlling the nictitating membrane response. The pairing of CS and UCS produces simultaneous activity in the pontine nucleus and the inferior olive nucleus and allows the CS to activate the longer neural circuit, eliciting the nictitating membrane response



concerns divergent results obtained from different measures of conditioning. SOP theory suggests that all response measures should yield a comparable indication of conditioning and that variations in the training conditions should have a similar effect on all response measures. Suppose that heart rate and eyeblink response are recorded during conditioning. Since both responses are assumed to reflect A2 neural activity, the optimal CS-UCS interval should be equal for both response measures. Yet, Vandercar and Schneiderman (1967) found maximum heart rate conditioning with a 2.25-second CS-UCS interval, while the strongest eyeblink response occurred with a 7.5-second CS-UCS interval.

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Recall our earlier discussion of the Thompson et al. (1984) study. We learned that destruction of the indirect inferior olive-cerebellar-red nucleus pathway eliminated the conditioned eyeblink response. However, the authors reported that this same surgical procedure had no effect on a conditioned heart rate response. To address these inconsistent findings, Wagner and Brandon modified SOP theory; we will look at this revision next.

Affective Extension of SOP, or AESOP

Wagner and Brandon (Brandon & Wagner, 1991; Brandon et al., 2002; Wagner & Brandon, 1989, 2001) suggested there are two distinct unconditioned response sequences—a sensory sequence and an emotive one. The sensory and emotive attributes of an unconditioned stimulus activate separate sequences of A1 and A2 activity. Further, the latency of the sensory and emotive activity sequences (A1 & A2) can differ; that is, A2 may take longer to develop for one component than the other. This difference leads to different optimal CS-UCS intervals for the emotive and sensory components. For example, a shorter-latency A2 activity for the sensory than emotive component of a UCS causes a shorter optimal CS-UCS interval for the sensory than the emotive CR. The differences in latencies between sensory and emotive A2 responses can result in one CS eliciting an emotive CR and another CS eliciting a sensory CR.

Affective extension of SOP theory (AESOP) has several additional aspects. A conditioned stimulus may activate a strong sensory conditioned response but only a weak emotive CR, or vice versa. This difference would explain the lack of correspondence between response measures of conditioning. Further, while the sensory A2 neural activity elicits a discrete response, the emotive A2 neural activity produces a diffuse reaction. For example, the sensory CR might be an eyeblink response, while the emotive CR could be a startle response. Finally, two unconditioned stimuli might activate the same emotive A2 activity but different sensory A2 activities. This would lead to both similarities and differences in the responses that separate UCSs condition.

Consider two studies that support AESOP theory. Tait and Saladin (1986) trained rabbits to respond to a 1,000-msec tone CS by presenting the tone 5,000-msec after a 100-msec shock UCS to the rabbits' eyes. Two conditioned response measures were taken in this study: the tendency of the CS to (1) suppress ongoing drinking behavior (emotive CR) and (2) elicit the eyeblink response (sensory CR). Tait and Saladin found a strong emotive CR; that is, the CS suppressed drinking. In contrast, the CS did not elicit the eyeblink response. In fact, the CS inhibited an eyeblink response to another CS, a common result with backward conditioning. Why did the rabbits acquire an emotive CR, but not a sensory CR? AESOP theory proposes that the CS occurred prior to the emotive A2 response but after the sensory A2 response.

A study by Betts, Brandon, and Wagner (1996) provides additional support for AESOP theory. These researchers paired a vibratory stimulus with a shock UCS in the first phase of an eyeblink conditioning study in rabbits. In the second phase, the researchers presented a tone, the vibratory stimulus used in the first stage, and a shock UCS. (Recall from Chapter 4 that this is using a blocking paradigm and that the presence of the vibratory stimulus should block conditioning to the tone.) The key variable in this study was whether the UCS in the

second phase was presented to the same or to a different eye than was used in the first phase. Betts, Brandon, and Wagner reported that both a reduced startle reaction and a reduced eyeblink response were conditioned to the tone when the UCS was presented to the same eye in both phases of the study. In contrast, when the UCS was presented to different eyes in each phase, the startle response to the tone was reduced, but the eyeblink response was equal to that elicited by the vibratory stimulus. Why did blocking occur to the startle response even when the location of the UCS changed, while changing the UCS location eliminated the blocking of the eyeblink response? According to Betts, Brandon, and Wagner, the startle response reflects the association of a stimulus and its emotional aspects, while the eyeblink response reflects an association between a stimulus and its sensory aspects. Changing the location of the UCS eliminated blocking of the eyeblink response because the sensory aspects of the UCS change with a change in UCS location, but this change had no effect on the startle response because the emotive aspects of the UCS do not change with a change in location.

BEFORE YOU GO ON

- Was Jared's response to Felicia's drinking different from or similar to his response to her hostility?
- Would Clarence become fearful of Felicia's drinking if it followed her hostility?

SECTION REVIEW

- Pavlov suggested that the simultaneous activity of CS and UCS brain areas creates a neural pathway between the CS and UCS brain centers, which allows the CS to elicit the UCR because of its ability to arouse the UCS and UCR brain areas.
- Siegel found that the UCR to morphine is analgesia, or reduced sensitivity to pain; the CR is hyperalgesia, or an increased sensitivity to pain; the UCR to insulin is hypoglycemia, the CR is hyperglycemia; the UCR to alcohol is hypothermia, the CR is hyperthermia.
- Siegel suggested that the conditioning of the opponent CR contributes to drug tolerance.
- Sometimes-opponent-process (SOP) theory suggests that the UCS elicits two unconditioned responses—a primary A1 component and a secondary A2 component.
- The primary A1 component is elicited rapidly by the UCS and decays quickly after the UCS ends. In contrast, the onset and decay of the secondary A2 component is gradual.
- SOP theory assumes that the secondary A2 component becomes the CR.
- If the A1 and A2 components differ, the CR will seem different from the UCR, while the CR will appear to be similar to the UCR when the A1 and A2 components are similar.
- AESOP proposes that the UCS elicits separate emotive and sensory unconditioned responses.
- According to AESOP, the emotive and sensory UCRs can have different time courses, which can lead to divergent conditioning outcomes for sensory and emotive CRs.

The Nature of the Pavlovian Conditioning Process

In Chapter 4, we learned that the predictiveness of the conditioned stimulus influences how readily a subject acquires a conditioned response. We also discovered that the predictive value of other stimuli also affects conditioning to the CS. How does a subject judge the relative predictiveness of a stimulus? Psychologists have developed several different views to explain the mechanism by which predictiveness affects classical conditioning (see Table 5.1). The Rescorla-Wagner associative view suggests that the availability of associative strength determines whether a CR develops to a CS paired with the UCS; comparator theory argues that performance of a conditioned response involves a comparison of the response strength to the CS and to competing stimuli; Mackintosh's attentional theory proposes that the relevance of and attention to a stimulus determine whether that stimulus will become associated with the UCS; and Baker's retrospective processing approach suggests that conditioning involves the continuous monitoring of contingencies between a CS and UCS, with the recognition of a lack of predictiveness diminishing the value of the CS.

The Rescorla-Wagner theory was developed to explain the influence of predictiveness on conditioning. We begin our discussion of the nature of classical conditioning with a description of this theory. We will then examine several Pavlovian conditioning phenomena that have been used to test the validity of the **Rescorla-Wagner associative model**. Some of this research has supported this theory, while other studies have pointed to its weaknesses. We will then look at alternatives to the Rescorla-Wagner associative model of conditioning.

Rescorla-Wagner Associative Model

The associative model of Pavlovian conditioning that Robert Rescorla and Allan Wagner (1972) developed expresses four main ideas. First, there is a maximum associative strength that can develop between a CS and UCS. The UCS determines the limit of associative strength; different UCSs support different maximum levels of conditioning, and therefore have different asymptotic values. Second, while the associative strength increases with each training trial, the amount of associative strength gained on a particular training trial depends on the level of prior training. Since the typical learning curve in Pavlovian conditioning is negatively accelerating (see Figure 5.6), more associative strength will accrue on early training trials than on later trials. In fact, as Figure 5.6 indicates, the increment on each conditioning trial declines with each CS-UCS pairing. Third, the rate of conditioning varies depending on the CS and the UCS used. Associative strength accrues quickly to some stimuli, but slowly to others. Figure 5.6 shows the learning curve of two stimuli: One stimulus readily gains associative strength, while conditioning to the other stimulus occurs slowly. Further, some UCSs produce more rapid learning than other UCSs. Fourth, the level of conditioning on a particular trial is influenced not only by the amount of prior conditioning to the stimulus, but also by the level of previous conditioning to other stimuli also paired with the UCS. A particular UCS can only support a certain amount of conditioning, even when more than one stimulus is paired with the UCS. When two (or more) stimuli are presented, these stimuli must share the associative strength the UCS can support. Thus, associative strength that accrues to one stimulus is not available to be conditioned to the other stimulus. For example, suppose two stimuli are paired with a UCS, and the maximum associative

TABLE 5.1 Explanation of Three Conditioning Phenomena by Four Models of Pavlovian Conditioning

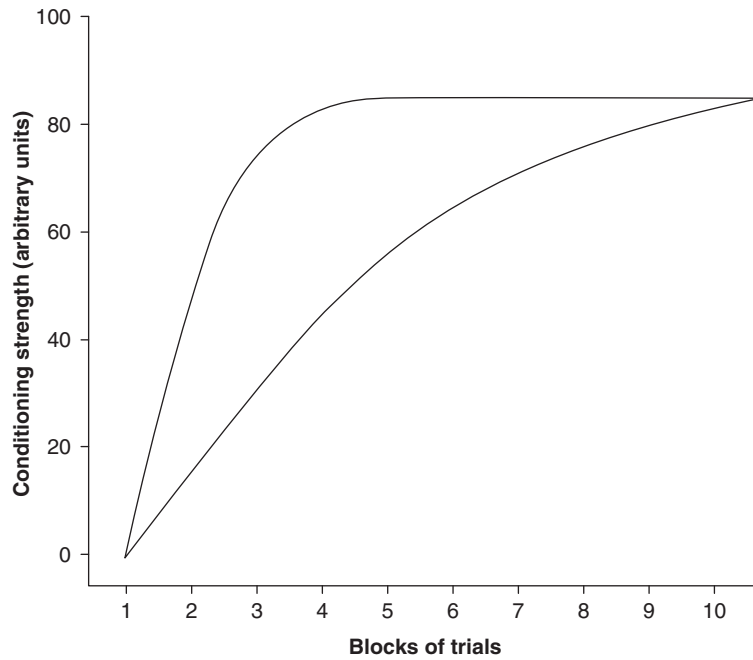
<i>Model or Theory</i>	<i>Overshadowing</i>	<i>Blocking</i>	<i>Predictiveness</i>
Rescorla-Wagner Model	Salient stimulus acquires associative strength more readily than nonsalient stimulus	Associative strength to blocking stimulus prevents conditioning to blocked stimulus	Context associations prevent conditioning to conditioned stimulus
Comparator Theory	Conditioning to salient stimulus is stronger than to nonsalient stimulus	Conditioning stronger for blocking stimulus than for blocked stimulus	Context associations stronger than conditioning to conditioned stimulus
Attentional Theory	Salient stimulus is more associable than nonsalient stimulus	Absence of surprise prevents conditioning to blocked stimulus	Animals learn that conditioned stimulus does not reliably predict unconditioned stimulus
Retrospective Processing Theory	Animals recognize the salience of different stimuli	Animals recognize greater contingency between blocking and unconditioned stimuli	Animals recognize the lack of contingency between conditioned stimulus and unconditioned stimulus

strength that the UCS can support is 10 units. If 7 units are conditioned to one cue paired with the UCS, only 3 units can develop to the other cue.

Rescorla and Wagner (1972) developed a mathematical equation based on the four ideas just outlined. Their mathematical model of Pavlovian conditioning is $V_A = K(\lambda - V_{AX})$. In this formula, V_A is the associative strength between the conditioned stimulus A and the UCS, and the ΔV_A is the change in associative strength that develops on a specific trial when the CS_A and the UCS are paired. The symbol K refers to the rate of conditioning determined by the nature of the CS_A and the intensity of the UCS. (The K value can be separated into α , or alpha, which refers to the power of CS_A , and β , or beta, which reflects the intensity of the UCS.) The symbol λ defines the maximum level of conditioning the UCS supports. The term V_{AX} indicates the level of conditioning that has already accrued

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FIGURE 5.6 The change in associative strength during conditioning for two different stimuli. One stimulus rapidly develops associative strength; the other acquires associative strength more slowly



to the conditioned stimulus (A) as well as to other stimuli (X) present during conditioning. Thus, $V_{AX} = V_A + V_X$.

To see how this mathematical model works, suppose that a light stimulus is paired with shock on five trials. Prior to training, the value of K is .5, λ is 90 and $V_A = 0$. When we apply these values to the Rescorla-Wagner model, we get:

Trial 1: $\Delta V_A = .5 (90 - 0) = 45$
Trial 2: $\Delta V_A = .5 (90 - 45) = 22.5$
Trial 3: $\Delta V_A = .5 (90 - 67.5) = 11.25$
Trial 4: $\Delta V_A = .5 (90 - 78.8) = 5.6$
Trial 5: $\Delta V_A = .5 (90 - 84.4) = 2.8$
Total Associative Strength after 5 Trials = 87.2

The data in this example show that conditioning to CS_A occurs rapidly; associative strength grows 45 units on Trial 1, 22.5 units on Trial 2, 11.25 units on Trial 3, 5.6 units on Trial 4, and 2.8 units on Trial 5. Thus, 87.2 units of associative strength accrued to the CS_A after just five trials of conditioning. The rapid development of associative strength indicates that CS_A is an intense and/or a salient stimulus or that the UCS is a strong stimulus, or both.

The Rescorla-Wagner model has been used to explain a number of conditioning phenomena. Let's see how it explains blocking (see Chapter 4). Suppose that we pair a light with a shock for five trials. The K value for the light is .5 and the maximum level of conditioning, or λ , is 90 units of associative strength. As we learned earlier, 87.2 units of associative strength would accrue to the light after five pairings with the shock. Next we pair the light, tone, and shock for five more trials. The K value for the tone is .5, and we would expect that five pairings of the tone and shock would yield strong conditioning. However, only 2.8 units of associative strength are still available to be conditioned, according to the Rescorla-Wagner model. And the tone must share this associative strength with the light cue. Because strong conditioning has already occurred to the light, the Rescorla-Wagner equation predicts little conditioning to the tone. The weak conditioning to the tone due to the prior accrued associative strength to light is illustrated in the calculations below:

Trial 6: $\Delta V_{\text{light}} = .5 (90 - 87.2) = 1.4$	$\Delta V_{\text{tone}} = .5 (90 - 87.2) = 1.4$
Trial 7: $\Delta V_{\text{light}} = .5 (90 - 90) = 0$	$\Delta V_{\text{tone}} = .5 (90 - 90) = 0$
Trial 8: $\Delta V_{\text{light}} = .5 (90 - 90) = 0$	$\Delta V_{\text{tone}} = .5 (90 - 90) = 0$
Trial 9: $\Delta V_{\text{light}} = .5 (90 - 90) = 0$	$\Delta V_{\text{tone}} = .5 (90 - 90) = 0$
Trial 10: $\Delta V_{\text{light}} = .5 (90 - 90) = 0$	$\Delta V_{\text{tone}} = .5 (90 - 90) = 0$
Total Associative Strength of light = 88.6	Total Associative Strength of tone = 1.4

We learned earlier that blocking occurs when a stimulus previously paired with a UCS is presented with a new stimulus and the UCS. The Rescorla-Wagner model suggests that blocking occurs because the initial CS has already accrued most or all of the associative strength, and little is left to condition to the other stimulus. As the previous equations show, little conditioning occurred to the tone because most of the associative strength had been conditioned to the light prior to the compound pairing of light, tone, and shock. Based on this explanation, the equation the Rescorla-Wagner model generates predicts cue blocking.

An Evaluation of the Rescorla-Wagner Model. Many studies have evaluated the validity of the Rescorla-Wagner model of Pavlovian conditioning. While many of these studies have supported this view, other observations have not been consistent with the Rescorla-Wagner model. We will first discuss one area of research—the UCS preexposure effect—that supports the Rescorla-Wagner view. Next, we will describe three areas of research—potentiation,

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CS preexposure, and cue deflation—that provide findings the Rescorla-Wagner model does not predict. Finally, we will discuss several alternative views of Pavlovian conditioning.

Suppose you have had several bouts of the flu recently and again become sick after eating a distinctive food. Would you develop an aversion to this food? Your previous experiences with sickness, independent of the particular food, probably would prevent the conditioning of an association between eating this food and being sick.

This example illustrates the effect of preexposure to the UCS (illness) without the CS (food) on the acquisition of a CR (aversion) when the CS is later presented with the UCS. Psychologists refer to this phenomenon as the **UCS preexposure effect**. Many studies have consistently observed that preexposure to the UCS impairs subsequent conditioning; for example, several researchers (Ford & Riley, 1984; Mikulka, Leard, & Klein, 1977) have demonstrated that the presentation of a drug that induces illness (UCS) prior to conditioning impairs the subsequent association of a distinctive food (CS) with illness. Similar preexposure interference has been reported with other UCSs (shock: Baker, Mercier, Gabel, & Baker, 1981; and food: Balsam & Schwartz, 1981).

Why does preexposure to the UCS impair subsequent conditioning? The Rescorla-Wagner model provides an explanation: The presentation of the UCS without the CS occurs in a specific environment or context, which results in the development of associative strength to the context.

Since the UCS can only support a limited amount of associative strength, conditioning of associative strength to the stimulus context reduces the level of possible conditioning to the CS. Thus, the presence of the stimulus context will block the acquisition of a CR to the CS when the CS is presented with the UCS in the stimulus context. (Referring back to blocking described in Chapter 4, it would be helpful to think of the context as CS_1 and the new stimulus as CS_2 .)

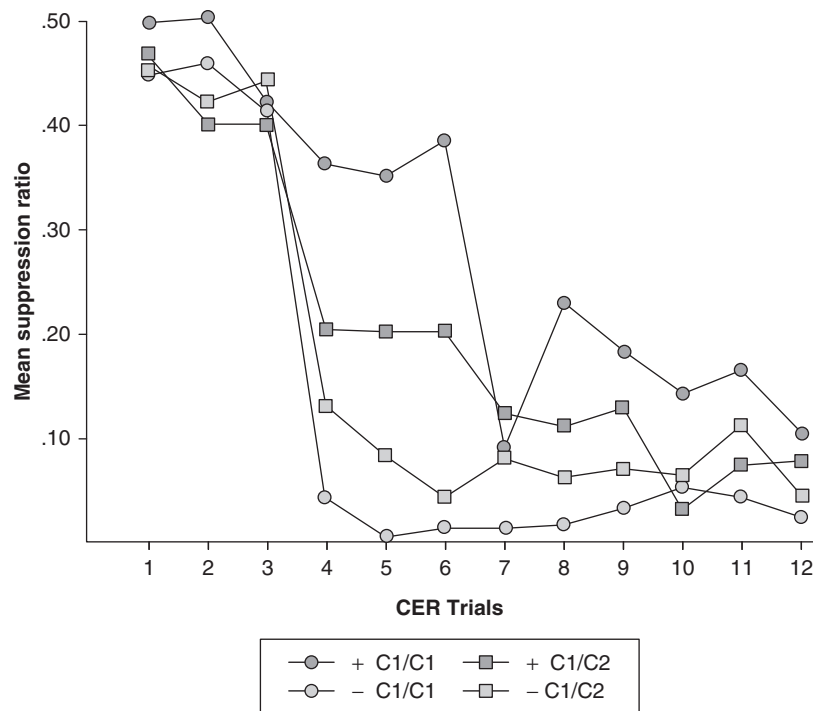
How can one validate the **context blocking** explanation of the UCS preexposure effect? One method is to change the context when the CS is paired with the UCS. Randich and Ross (1985) found that the UCS preexposure effect was attenuated when the preexposure context (noise from a fan, but no light, painted walls, or the odor of Pine-Sol) was different from the conditioning context (light, black-and-white-striped walls, and Pine-Sol odor, but no fan) presented prior to noise CS and shock pairings (see Figure 5.7). As a result of the change in context, no stimuli were present during conditioning that could compete with the association of the CS and the UCS. Thus, the CR was readily conditioned to the CS when paired with the UCS in the new context.

Other researchers also have reported that context change attenuates the UCS preexposure effect. Hinson (1982) found that the effect of UCS preexposure on eyelid conditioning in the rabbit was attenuated by a change in contextual stimuli between preexposure and eyelid conditioning, while de Brugada, Hall, and Symonds (2004) reported the UCS preexposure effect was reduced when lithium chloride was injected during preexposure and orally consumed during flavor aversion conditioning. These results strongly suggest that contextual associations formed during UCS preexposure are responsible for the decrease in the conditioning to a CS paired with a UCS.

In the next three sections, we will discuss several findings that the Rescorla-Wagner model does not predict. The first problem area is the potentiation effect.

The Potentiation of a Conditioned Response. The Rescorla-Wagner model predicts that when a salient and a nonsalient cue are presented together with the UCS, the salient cue will

FIGURE 5.7 The influence of context change on the UCS preexposure effect. Animals in the +C1/C1 experimental group, which received both preexposure and conditioning in Context 1, showed significantly slower acquisition of a conditioned emotional response than did animals in the +C1/C2 experimental group (given preexposure in Context 1 and conditioning in Context 2). Control animals in the -C1/C1 and -C1/C2 groups who did not receive UCS preexposure readily conditioned fear to either Context 1 or 2



accrue more associative strength than the nonsalient cue. This phenomenon, called **overshadowing**, was originally observed by Pavlov (1927). Pavlov found that a more intense tone overshadowed the development of an association between a less intense tone and the UCS. Overshadowing is readily observed in other situations. For example, Lindsey and Best (1973) presented two novel fluids (saccharin and casein hydrosylate) prior to illness. They found that a strong aversion developed to the salient saccharin flavor, but only a weak aversion developed to the less salient casein hydrosylate solution.

Overshadowing does not always occur when two cues of different salience are paired with a UCS; in fact, in some circumstances the presence of a salient cue produces a stronger CR than would have occurred had the less salient cue been presented alone with

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the UCS. The increased CR to a less salient stimulus because of the simultaneous pairing of a more salient cue during conditioning was first described by John Garcia and his associates (Garcia & Rusiniak, 1980; Rusiniak, Palmerino, & Garcia, 1982). They observed that the presence of a salient flavor cue potentiated rather than overshadowed the establishment of an aversion to a less salient odor cue paired with illness.

Why does the presence of a salient taste cue potentiate rather than overshadow the acquisition of an odor aversion? According to Garcia and Rusiniak (1980), the taste stimulus “indexes” the odor as a food cue, and thereby mediates the establishment of a strong odor aversion. This indexing has considerable adaptive value. The taste cue’s **potentiation** of the odor aversion enables an animal to recognize a potentially poisonous food early in the ingestive sequence. Thus, an odor aversion causes animals to avoid dangerous foods before even tasting them.

Rescorla (1982) presents a different view of the potentiation effect, a view consistent with the Rescorla-Wagner model. According to Rescorla, potentiation occurs because an animal perceives the compound stimuli (taste and odor) as a single unitary event and then mistakes each individual element for the compound. If Rescorla’s view is accurate, the potentiation effect should depend upon the strength of the taste-illness association. Potentiation should occur with a strong taste aversion, and weakening of the taste-illness association should result in an elimination of the potentiation effect. Rescorla (1981) presented evidence to support his view; that is, he found that extinction of the taste aversion also attenuated the animal’s aversion to an odor cue. However, Lett (1982) observed that taste-alone exposure eliminated the taste aversion but not the odor aversion. The cause of potentiation thus seems unclear; we will discuss this phenomenon again when we look at Rescorla’s within-compound view later in the chapter.

The CS Preexposure Effect. Recall our discussion of Clarence’s failure to associate Felicia’s drinking and aggression in the chapter opening vignette. The **CS preexposure effect** provides an explanation for his failure to develop apprehension about Felicia’s drinking. Many studies (Bonardi & Yann Ong, 2003) have reported that preexposure to a specific stimulus (drinking) subsequently retarded the development of a CR (apprehension) to that stimulus when paired with a UCS (hostility). The CS preexposure effect has been reported in a variety of Pavlovian conditioning situations, including conditioned water licking in rats (Baker & Mackintosh, 1979), conditioned fear in rats (Pearce, Kaye, & Hall, 1982) and humans (Booth, Siddle, & Bond, 1989), eyelid conditioning in rabbits (Rudy, 1994), leg-flexion conditioning in sheep and goats (Lubow & Moore, 1959), and flavor aversion learning in rats (Fenwick, Mikulka, & Klein, 1975).

Why is the CS preexposure effect a problem for the Rescorla-Wagner model? According to Rescorla and Wagner (1972), exposure to the CS prior to conditioning should have no effect on the subsequent association of the CS with the UCS. This prediction is based on the assumption that the readiness of a stimulus to be associated with a UCS depends only on the intensity and salience of the CS; the parameter K represents these values in the Rescorla-Wagner model. While neither the intensity nor the salience of the CS is changed as the result of CS preexposure, the subsequent interference with conditioning indicates that the associability of the CS changes when the CS is experienced without the UCS prior to conditioning.

How can we explain the influence of CS preexposure on subsequent conditioning? One explanation involves modifying the Rescorla-Wagner model to allow for a change in the value of K as the result of experience. Yet, the effect of CS preexposure on the acquisition

of a CR appears to involve more than just a reduction in the value of K . Instead, Mackintosh (1983) argues that animals learn that a particular stimulus is irrelevant when it predicts no significant event; stimulus irrelevance causes the animal to ignore that stimulus in the future. This failure to attend to the CS and the events that follow it may well be responsible for the interference with conditioning that CS preexposure produces. We will look more closely at this attentional view of CS preexposure when we describe Mackintosh's attentional model of conditioning.

The Cue Deflation Effect. The Rescorla-Wagner model suggests that the overshadowing phenomenon involves greater conditioning to a more salient rather than a less salient stimulus; that is, greater associative strength accrues to the more salient rather than less salient cue. What do you suppose would happen to an animal's response to the less salient stimulus if the conditioned response to the more salient stimulus were extinguished? The Rescorla-Wagner model does not suggest any change in the reaction to the less salient cue. However, a number of studies (Kaufman & Bolles, 1981; Matzel, Schachtman, & Miller, 1985) reported that extinction of the more salient (or overshadowing) stimulus increased the response to the less salient (or overshadowed) stimulus. Not all studies find a **cue deflation effect**, or increased responding to the less salient stimulus, following extinction to a more salient cue; instead, some studies report a decreased response to both the overshadowing and overshadowed stimuli (Durlach, 1989).

An increased response to a CS without additional experience also occurs with the extinction of context associations acquired with UCS preexposure. Recall that exposure to the UCS prior to CS-UCS pairings produces a weaker response to the CS than when no UCS preexposure is given.

We learned earlier that context-UCS associations acquired during UCS preexposure block strong conditioning to the CS. Several studies (Matzel, Brown, & Miller, 1987; Timberlake, 1986) show that postconditioning extinction of the response to the training context results in an enhanced response to the CS. Again, not all studies that extinguish the response to the training context have noted an increased responding to the CS (refer to Durlach, 1989). What process is responsible for the change in response to one stimulus following the extinction of a response to another stimulus? Why do some studies report that diminished responding to one stimulus increased the response to the CS, while other studies find that this same procedure decreased the reaction to the CS? The next two sections address both of these questions.

The Importance of Within-Compound Associations

Suppose that a tone and light are paired together with food. According to the Rescorla-Wagner model, the light and tone will compete for associative strength. More recently, Rescorla (Rescorla & Durlach, 1981) suggested that rather than two stimuli competing for associative strength, a **within-compound association** can be established between the light and tone during conditioning. This within-compound association will result in a single level of conditioning to both stimuli. One procedure facilitating a within-compound association is the simultaneous pairing of both stimuli. As a result of developing a within-compound association, any change in the value of one stimulus will have a similar impact on the other stimulus.

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We described earlier the observation that the presence of a salient taste cue potentiated the aversion to a nonsalient odor cue. Garcia and Rusiniak (1980) suggested that the taste stimulus “indexes” the odor stimulus as a food cue and thereby mediates the establishment of a strong odor aversion. If this view were accurate, then a taste could potentiate an aversion to an odor, but an odor would not potentiate an aversion to a taste cue. However, Batsell, Trost, Cochran, Blankenship, and Batson (2003) observed that an odor can potentiate the aversion to a taste, a result suggesting that the taste and odor association is symmetrical. We also learned earlier that Rescorla explains the potentiation phenomenon by suggesting that the within-compound association of a salient taste cue and a nonsalient odor cue leads to a strong aversion (potentiation) when both cues were paired with illness. Batsell et al.’s (2003) observation that a taste and odor association is symmetrical provides support for Rescorla’s within-compound explanation of potentiation.

The presence of a taste cue potentiates not only an odor aversion, but also a contextual aversion (Boakes, Westbrook, & Barnes, 1992). Boakes, Westbrook, and Barnes observed that the pairing of a taste in a distinctive context with illness potentiated the aversion to that context, measured by the intake of another taste, when compared to a condition in which the context was associated alone with illness.

The within-compound conditioning view suggests that potentiation is dependent upon the establishment of an association between the odor and taste cues. According to this view, the failure to form a within-compound association between odor and flavor should eliminate potentiation. One procedure used to prevent within-compound associations is pairing the odor and taste cues sequentially rather than simultaneously. This procedure eliminates the flavor stimulus’s potentiation of the odor cue (Davis, Best, & Grover, 1988; Holder & Garcia, 1987).

While within-compound conditioning may contribute to potentiation, it is not the entire story. As we learned earlier, Lett (1982) did not find that extinction of the taste aversion eliminated the odor aversion. Further, a number of studies (Bouton, Jones, McPhillips, & Swartzentruber, 1986; Schneider & Pinnow, 1994) have found that a taste cue overshadows rather than potentiates an odor aversion. In these studies, overshadowing occurred under conditions that were favorable to within-compound associations.

BEFORE YOU GO ON

- How would the Rescorla-Wagner model explain Clarence’s failure to develop an association between Felicia’s drinking and her hostility?
- How would the Rescorla-Wagner model explain Jared’s association between Felicia’s drinking and her hostility?

SECTION REVIEW

- The Rescorla-Wagner model proposes (1) that there is a maximum level of conditioning supported by the UCS; (2) the associative strength increases readily early in training but more slowly later in conditioning; (3) the rate of conditioning is more rapid with some CSs or UCSs than with others; and (4) the level of conditioning on a particular trial depends upon the level of prior conditioning to the CS and to the other stimuli present during conditioning.

- According to the Rescorla-Wagner theory, blocking occurs as a result of conditioning associative strength to one stimulus, thereby preventing conditioning to a second stimulus due to a lack of available associative strength.
- Preexposure to the UCS impairs subsequent conditioning due to contextual blocking; a change in context eliminates the UCS preexposure effect.
- In a compound conditioning situation, overshadowing occurs when the presence of a salient stimulus prevents conditioning to a less salient stimulus, while potentiation occurs when the presence of a salient stimulus enhances the conditioning to the less salient stimulus.
- Rescorla suggests that under some conditions two stimuli paired with a UCS develop a within-compound association (potentiation) instead of competing for associative strength (overshadowing).

Recall our discussion of the cue deflation effect, presented in the previous section. We learned that several studies showed that the extinction of a response to one component of a compound stimulus enhanced the response to the other component. Yet, other experiments reported that extinction to one component also reduced response to the other component. The latter result is consistent with the within-compound analysis; that is, a within-compound association is established to both components. Consequently, reducing the response to one has a comparable effect on the other. However, the former studies are not consistent with the within-compound analysis. Comparator theory offers an explanation why the extinction of a response to one component would increase the response to the other.

A Comparator Theory of Pavlovian Conditioning

Ralph Miller and his associates (Denniston, Savastano, Blaisdell, & Miller, 2003; Denniston, Savastano, & Miller, 2001; Miller & Matzel, 1989; Urcelay & Miller, 2006) have proposed that animals learn about all CS-UCS relationships. However, a particular association may not be evident in the animal's behavior. A strong CS-UCS association may exist but not be expressed in behavior, when compared with another CS even more strongly associated with the UCS. Thus, the ability of a particular stimulus to elicit a CR depends upon its level of conditioning compared to other stimuli. Only when the level of conditioning to that stimulus exceeds that of other stimuli will that CS elicit the CR.

Consider the blocking phenomenon to illustrate **comparator theory**. The comparator approach proposes that an association may exist between CS_2 and the UCS but not be evident in terms of response because of the stronger CS_1 -UCS association. (Recall that the Rescorla-Wagner model assumes that the presence of the CS_1 blocks or prevents the establishment of the CS_2 -UCS association.) The comparator theory suggests that there is one condition in which the CS_2 can elicit the CR in a blocking paradigm. The extinction of the conditioned response to the CS_1 can allow the CS_2 to now elicit the CR. The reason that extinction of the response to the CS_1 results in response to the CS_2 is that the comparison now favors the CS_2 -UCS association. Prior to extinction, the CS_1 -UCS association was stronger than the CS_2 -UCS association. After extinction, the CS_2 -UCS association is stronger than CS_1 -UCS association.

We learned in an earlier section that extinction of response to the training context eliminated the UCS preexposure effect. These studies (Matzel et al., 1987; Timberlake, 1986)

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show that deflation of response to the training context leads to an increased response to the CS. This greater response occurred even though no additional CS-UCS pairings were given. Additional support for the comparator theory comes from experiments in which devaluation of the overshadowing stimulus caused an increased response to the overshadowed stimulus (Kaufman & Bolles, 1981; Matzel et al., 1985). This observation suggests that an association between the overshadowed stimulus and the UCS did form, but it was not evident because of its unfavorable comparison with an overshadowing stimulus.

While these observations provide support for the comparator theory, not all studies have found that the deflation of one stimulus increases the response to another stimulus. In fact, many studies have reported that extinguishing the response to one stimulus produces a comparable reduction in the other stimulus, a result that favors the within-compound associative view presented in the previous section.

What is responsible for this discrepancy in results? Durlach (1989) suggested that the presence of strong within-compound associations might overwhelm the comparator effect. In her view, the comparator effect would only be evident when within-compound associations are weak.

However, Blaisdell, Gunther, and Miller (1999) reported that the amount of posttraining extinction (deflation) is the critical variable that determines whether the CS elicits the CR. These researchers found that extensive extinction trials are needed to eliminate the conditioning to the comparator stimulus and increase the response to the CS.

So what causes the cue deflation effect? Van Hamme and Wasserman (1994) modified the Rescorla-Wagner theory to account for the cue deflation effect. In their view, extinguishing the response to one conditioned stimulus (the deflated stimulus) changes the value of K to a second conditioned stimulus, which serves to increase the associative strength of the second conditioned stimulus (see Van Hamme & Wasserman, 1994 for the mathematical revision of the Rescorla-Wagner associative model that explains the cue deflation effect). Denniston et al. (2001) have presented several research findings that this modified associative theory cannot explain but that the comparator theory can. For example, Denniston et al. reported that extinction of a second-order comparator stimulus (or a comparator stimulus for the comparator stimulus) increases the response to the first-order comparator stimulus, which then serves to decrease the response to the CS. Future research is needed to clarify the processes responsible for the cue deflation effect, as well as evaluate the validity of the associative and comparator theories of Pavlovian conditioning.

Mackintosh's Attentional View

Nicholas Mackintosh (1975) suggested that animals seek information from the environment that predicts the occurrence of biologically significant events (UCSs). Once an animal has identified a cue that reliably predicts a specific event, it ignores other stimuli that also provide information about the event. According to Mackintosh, animals attend to stimuli that are predictive and ignore those that are not essential. Thus, conditioning depends not only on the physical characteristics of stimuli, but also on the animal's recognition of the correlation (or lack of correlation) between events (CS & UCS).

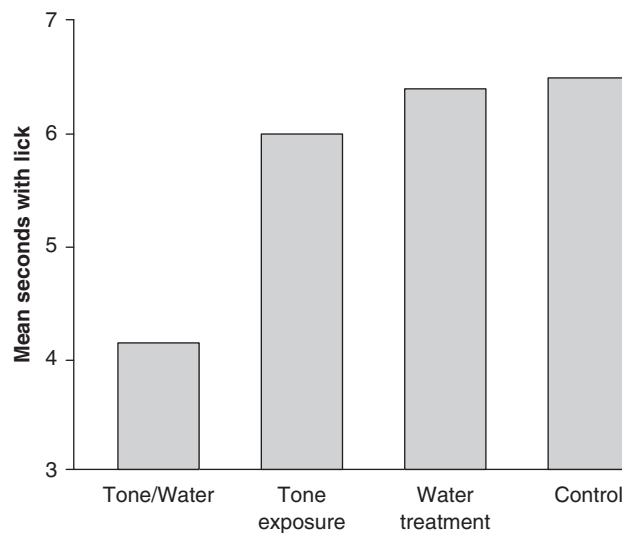
Mackintosh's attentional view of Pavlovian conditioning can explain the CS preexposure effect that poses a problem for the Rescorla-Wagner model. We discovered earlier in the

chapter that CS preexposure impairs the acquisition of the CR when the CS and UCS are later paired. According to Mackintosh, an animal learns that the CS is irrelevant as a result of preexposure to the CS. Once the animal discovers that a stimulus is irrelevant, it will stop attending to that stimulus and will have difficulty learning that the CS is correlated with the UCS.

Support for this **learned irrelevance** view of CS preexposure comes from studies in which uncorrelated presentations of the CS and UCS prior to conditioning led to substantial interference with the acquisition of the CR. In fact, Baker and Mackintosh (1977) found that uncorrelated presentations of the CS and the UCS produce significantly greater interference than did CS preexposure or UCS preexposure alone. In their study, the response of water licking to a tone was significantly less evident in animals receiving prior unpaired presentations of the tone (CS) and water (UCS) than either tone alone, water alone, or no preexposure (see Figure 5.8). This greater impairment of subsequent conditioning when the CS and UCS are unpaired also has been demonstrated in studies of conditioning fear in rats (Baker, 1976) and the eyeblink response in rabbits (Siegel & Domjan, 1971).

Uncorrelated CS and UCS preexposure not only impairs subsequent excitatory conditioning; it also impairs inhibitory conditioning (Bennett, Wills, Oakeshott, & Mackintosh, 2000). The fact that uncorrelated CS and UCS exposures impaired inhibitory as well as excitatory conditioning provides additional evidence for the learned irrelevance explanation of the CS preexposure effect.

FIGURE 5.8 The amount of licking to a tone (CS) paired with water (UCS) is significantly less in animals preexposed to both the tone and water than to only the water or only the tone, or with no preexposure



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Several studies by Geoffrey Hall and his associates (Hall & Channell, 1985; Hall & Honey, 1989) provide additional evidence for an attentional view of the CS preexposure effect. Animals exposed to a novel stimulus exhibit an orienting response to the novel stimulus. Hall and Channell (1985) showed that repeated exposure to light (CS) leads to habituation of the orienting response to that stimulus (see Chapter 3). They also found that later pairings of the light (CS) with milk (UCS) yield a reduced CR compared with control animals who did not experience preexposure to the light CS. These results suggest that habituation of an orienting response to a stimulus is associated with the later failure of conditioning to that stimulus.

What if the orienting response could be reinstated to the conditioned stimulus? Would this procedure restore conditionability to the stimulus? Hall and Channell (1985) reported that the presentation of the conditioned stimulus in a novel context reinstated the orienting response. They also found that pairing the CS and UCS in the new context led to a strong CR. These results indicate that a reinstatement of the orienting response eliminated the CS preexposure effect; that is, the CS now elicited a strong CR.

Why would reinstatement of the orienting response cause CS conditionability to return? An orienting response indicates that an animal is attending to the stimulus, and attention allows the stimulus to be associated with the UCS. These observations provide further support for an attentional view of the CS preexposure effect.

We learned earlier that uncorrelated CS and UCS exposures impaired both excitatory and inhibitory conditioning. Bonardi, Hall, and Ong (2005) reported that while uncorrelated CS and UCS exposures impaired excitatory conditioning, inhibitory conditioning was facilitated by uncorrelated CS and UCS presentations, a result that they attributed to latent learning (see Chapter 3) rather than learned irrelevance. The reasons for these different results remain to be investigated.

The Retrospective Processing View

Theories of Pavlovian conditioning have traditionally held that learning occurs at the time of training and that response is based upon the level of training. Baker and Mercier (1989) refer to these models of Pavlovian conditioning as input-based theories. The Rescorla-Wagner associative theory, Rescorla's within-compound association view, and Mackintosh's attentional perspective are input-based theories of Pavlovian conditioning. In contrast, Miller's comparative theory is an output-based model because it suggests that performance is determined by comparing the level of prior conditioning to each stimulus at the time of testing. However, all of these theories assume that unless further conditioning is provided, the level of learning remains constant after training.

Baker and Mercier (1989) present a very different view of Pavlovian conditioning. They contend that the level of conditioning to a CS can change even with no additional CS-UCS pairings. According to Baker and Mercier, animals constantly assess the contingencies between events in their environment. Rather than viewing learning as a static representation of the degree of correlation between events, these researchers suggest that learning changes over time as an animal encounters new information about the degree of contingency between a CS and UCS. For example, what may seem to be two highly correlated events may later be viewed as having little correlation. This change in learning would occur

if initial CS-UCS pairings were followed by many UCS-alone experiences. Baker and Mercier refer to the constant assessment of contingencies as **retrospective processing**. New data may cause an animal to reassess past experiences and form a new representation of the relationship between the CS and UCS.

Retrospective processing requires the ability to remember past experiences. It also assumes that an animal has a representation of past encounters that it can modify. In this section, we will look at several studies that support retrospective processing.

Suppose that after a tone and light were paired with a shock, only the tone was presented prior to the shock. How would the animal respond to the light? Baker and Baker (1985) performed such a study and found that fear of the light was reduced compared to a control group that did not receive tone-shock pairings. This study is similar to the blocking paradigm described in Chapter 4. In fact, the only difference between the procedures is the order of tone-light-shock and tone-shock pairings. Baker and Mercier (1989) refer to a procedure in which the tone-shock follows rather than precedes tone-light-shock pairing as **backward blocking**.

What causes backward blocking? Baker and Mercier (1989) argue that when animals receive tone-shock after tone-light-shock pairings, they discover that the tone is a better predictor of shock than the light. Through retrospective processing, the animals decide that the light is not an adequate predictor of shock. This decision causes the animal to no longer fear the light.

Recall our discussion of the UCS preexposure effect. We learned that exposure to the UCS impaired later conditioning of the CS and UCS. A similar impairment of conditioning occurs when UCS-alone experiences are intermixed with CS-UCS pairings (Jenkins, Barnes, & Barrera, 1981). According to Baker and Mercier (1989), the animal revises its view of the contingency between the CS and UCS as a result of UCS-alone experience. In other words, the animal retrospectively decides that the CS no longer correlates well with the UCS.

Miller and his colleagues (Denniston, Savastano, & Miller, 2001; Savastano, Escobar, & Miller, 2001) have reported that backward blocking does not always occur. These researchers found that if a CS has become able to produce “robust responding”—the CS consistently produces an intense CR—the CS appears to become “immune” to backward blocking, and additional training to a competing stimulus will not reduce the response to the CS. For example, if a CS is “inherently biologically significant” or acquires biological significance through conditioning, attempts to reduce response to the CS by pairing a competing (comparator) cue with the UCS will have little effect on response to the CS. You might wonder what the term *biological significance* means. We will leave you in suspense until Chapter 12.

BEFORE YOU GO ON

- How would Mackintosh’s attentional view explain the difference in Jared’s and Clarence’s responses to Felicia’s drinking?
- What prediction would Baker’s retrospective processing model make for the development of a drinking-hostility association following Clarence’s conversation with his friend Jared?

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SECTION REVIEW

- Comparator theory argues that animals learn about all CS-UCS relationships and blocking occurs when the animal does not respond to the CS₂ because the CS₂-UCS association is weaker than the CS₁-UCS association.
- Deflation of the value of the CS₁ by extinction results in an increased response to the CS₂ due to the favorable comparison of the CS₂-UCS association to the CS₁-UCS association.
- Mackintosh's attentional view suggests that animals seek information that predicts the occurrence of biologically significant events (UCSs).
- As the result of preexposure to the CS, an animal learns that the CS is irrelevant, which makes it difficult to later learn that the CS correlates with the UCS.
- Baker's retrospective processing theory proposes that animals are continuously monitoring the contingency between CS and UCS and that experience with a CS or UCS alone after conditioning can lead the animal to reevaluate the predictive value of the CS.
- The backward blocking phenomenon occurs when there is a reduced responding to the CS₂ when CS₁-UCS pairings follow CS₁-CS₂-UCS pairings.

We have described several very different theories of Pavlovian conditioning. In all likelihood, each theory accounts for some aspects of Pavlovian conditioning. Future research will clarify the precise contribution of each theory.

APPLICATIONS OF PAVLOVIAN CONDITIONING

We will discuss three applications of Pavlovian conditioning in this chapter. The first involves the use of Pavlovian conditioning principles to modify phobic behavior. This procedure, called systematic desensitization, has been used for over 40 years to eliminate the fears of people with phobias. The second application, which involves extinction of a person's craving for a drug, has only recently been used to treat drug addiction. The last application has not been clinically tested, but it represents a possible use of Pavlovian conditioning to correct immune system dysfunction in patients with lupus and other diseases of the immune system.

Systematic Desensitization

Suppose Ben is extremely frightened of taking examinations. This fear could cause Ben to do poorly in college. What can be done to allow Ben to take examinations with minimal or no fear? **Systematic desensitization**, a therapy developed by Joseph Wolpe, acts to inhibit fear and suppress phobic behavior (a **phobia** is an unrealistic fear of an object or situation). Wolpe's therapy can help people with extreme test anxiety. His treatment is based on Pavlovian conditioning principles and represents an important application of classical conditioning. Let's examine this technique to discover how Pavlovian conditioning can alleviate extreme fear.

Original Animal Studies

Wolpe's therapy developed from his animal research. In an initial study (Wolpe, 1958), he shocked one group of cats in their home cages after they heard a buzzer. For the other cats, he paired the buzzer with food in the home cages and then shocked them. Both groups of cats later showed extreme fear of the buzzer; one indication of their fear was their refusal to eat when hearing the buzzer. Since fear inhibited eating, Wolpe reasoned that eating could—if sufficiently intense—suppress fear. As we learned in Chapter 1, counterconditioning is the process of establishing a response that competes with a previously acquired response. Wolpe suggested that counterconditioning is a potentially effective way of treating human phobic behavior. He based this idea on three lines of evidence: (1) Sherrington's (1906) statement that an animal can only experience one emotional state at a time—a phenomenon Wolpe termed reciprocal inhibition; (2) Jones's (1924) report that she had successfully eliminated a young boy's conditioned fear of rabbits by presenting the feared stimulus (a rabbit) while the boy was eating (see Chapter 1); and (3) Wolpe's own research using cats.

Wolpe (1958) initially placed the cats—which had developed a conditioned fear of the buzzer and the environment in which the buzzer was experienced—in a cage with food; this cage was quite dissimilar to their home cage. He used the dissimilar cage, which produced only a low fear level due to little generalization (see Chapter 1), because the home cage would produce too intense a fear and therefore inhibit eating. Wolpe observed that the cats ate in the dissimilar cage and did not appear afraid either during or after eating. Wolpe concluded that in the dissimilar environment, the eating response had replaced the fear response. Once the fear in the dissimilar cage was eliminated, the cats were less fearful in another cage more closely resembling the home cage. The reason for this reduced fear was that the inhibition of fear conditioned to the dissimilar cage generalized to the second cage. Using the counterconditioning process with this second cage, Wolpe found that presentation of food in this cage quickly reversed the cats' fear. Wolpe continued the gradual counterconditioning treatment by slowly changing the characteristics of the test cage until the cats were able to eat in their home cages without any evidence of fear. Wolpe also found that a gradual exposure of the buzzer paired with food modified the cats' fear response to the buzzer.

Clinical Treatment

Wolpe (1958) believed that human phobias could be eliminated in a manner similar to the one he used with his cats. He chose not to use eating to inhibit human fears, but instead used three classes of inhibitors: relaxation, assertion, and sexual responses. We will limit our discussion in this chapter to the use of relaxation.

Wolpe's (1958) therapy using relaxation to counter human phobic behavior is called **systematic desensitization**. Basically, desensitization involves relaxing while first imagining and then experiencing anxiety-inducing scenes. To promote relaxation, Wolpe used a series of muscle exercises Jacobson developed in 1938. These exercises involve tensing a particular muscle and then releasing this tension. Presumably, tension is related to anxiety, and tension reduction is relaxing. The patient tenses and relaxes each major muscle group in a specific sequence.

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Relaxation is most effective when the tension phase lasts approximately 10 seconds and is followed by 10 to 15 seconds of relaxation for each muscle group. The typical procedure requires about 30 to 40 minutes to complete; however, later in therapy, patients need less time as they become more readily able to experience relaxation. Once relaxed, patients are required to think of a specific word (for example, *calm*). This procedure, which Russell and Sipich (1973) labeled **cue-controlled relaxation**, promotes the development of a conditioned relaxation response that enables a word cue to elicit relaxation promptly; the patient then uses the cue to inhibit any anxiety occurring during therapy.

The desensitization treatment consists of four separate stages: (1) the construction of the anxiety hierarchy; (2) relaxation training; (3) counterconditioning, or the pairing of relaxation with the feared stimulus; and (4) an assessment of whether the patient can successfully interact with the phobic object. In the first stage, patients are instructed to construct a graded series of anxiety-inducing scenes related to their phobia. A 10- to 15-item list of low-, moderate-, and high-anxiety scenes is typically employed. The patient writes descriptions of the scenes on index cards and then ranks them from those that produce low anxiety to those that produce high anxiety.

Paul (1969) identified two major types of hierarchies: thematic and spatial-temporal. In a **thematic hierarchy**, the scenes are related to a basic theme. Table 5.2 presents a hierarchy detailing the anxiety an insurance salesman experienced when anticipating interactions with coworkers or clients. Each scene in the hierarchy is somewhat different, but all are related to his fear of possible failure in professional situations. In contrast, a **spatial-temporal hierarchy** is based on phobic behavior in which the intensity of fear is determined by distance (either physical or temporal) to the phobic object. The test anxiety hierarchy shown in Table 5.3 indicates that the level of anxiety is related to the proximity to exam time.

We need to point out one important aspect of the hierarchy presented in Table 5.3. Perhaps contrary to your intuition, this student experienced more anxiety en route to the exam than when in the test area. Others may have a different hierarchy; when taking the exam, they experience the most fear. As each individual's phobic response is highly idiosyncratic and dependent on that person's unique learning experience, a hierarchy must be specially constructed for each person. Some phobias require a combination of thematic and spatial-temporal hierarchies. For example, a person with a height phobia can experience varying levels of anxiety at different places and at different distances from the edges of these places.

After the hierarchy is constructed, the patient learns to relax. Relaxation training follows the establishment of the hierarchy to prevent the generalization of relaxation to the hierarchical stimuli and thereby preclude an accurate assessment of the level of fear to each stimulus. The counterconditioning phase of treatment begins following relaxation training. The patient is instructed to relax and imagine as clearly as possible the lowest scene on the hierarchy. Since even this scene elicits some anxiety, Masters et al. (1987) suggested that the first exposure be quite brief (5 seconds). The duration of the imagined scene can then be slowly increased as counterconditioning progresses.

It is important that the patient not become anxious while picturing the scene; otherwise, additional anxiety, rather than relaxation, will be conditioned. The therapist instructs the patient to signal when experiencing anxiety, and the therapist terminates the scene. After

TABLE 5.2 Thematic Hierarchy

<i>Level</i>	<i>Scene</i>
1	In your office with an agent, R.C., discussing a prospective interview. The client in question is stalling on his payment, and you must tell R.C. what to do.
2	Monday morning working in your office. In a few minutes you will attend the regularly scheduled sales meeting. You are prepared for the meeting.
3	Conducting an exploratory interview with a prospective client.
4	Sitting at home. The telephone rings.
5	Anticipating returning a call from the district director.
6	Anticipating returning a call from a stranger.
7	Entering the Monday sales meeting unprepared.
8	Anticipating a visit from the regional director.
9	Listening as a fellow agent requests a joint visit with a client.
10	On a joint visit with a fellow agent.
11	Attempting to close a sale.
12	Thinking about attending an agents' and managers' meeting.
13	Thinking of contacting a client who should have been contacted earlier.
14	Thinking about calling a prospective client.
15	Thinking about the regional director's request for names of prospective agents.
16	Alone, driving to prospective client's home.
17	Calling a prospective client.

Note: In the fear hierarchy, a higher level represents greater fear.

a scene has ended, the patient is instructed to relax. The scene can again be visualized when relaxation has been reinstated. If the individual can imagine the first scene without any discomfort, the next scene in the hierarchy is imagined. Counterconditioning at each level of the hierarchy continues until the patient can imagine the most aversive scene without becoming anxious.

Clinical Effectiveness

The last phase of desensitization evaluates the therapy's success. To test the effectiveness of desensitization, the individual must encounter the feared object. The success of desensitization

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TABLE 5.3 Spatial-Temporal Hierarchy	
<i>Level</i>	<i>Scene</i>
1	Four days before an examination.
2	Three days before an examination.
3	Two days before an examination.
4	One day before an examination.
5	The night before an examination.
6	The examination paper lies face down before the student.
7	Awaiting the distribution of examination papers.
8	Before the unopened doors of the examination room.
9	In the process of answering an examination paper.
10	On the way to the university on the day of the examination.

Note: In the fear hierarchy, a higher level represents greater fear.

as a treatment for phobic behavior is quite impressive. Wolpe (1958) reported that 90 % of 210 patients showed significant improvement with desensitization, compared to a 60 % success rate when psychoanalysis was used. The comparison is more striking when one considers that desensitization produced a rapid extinction of phobic behavior—according to Wolpe (1976), a range of 12 to 29 sessions was effective—compared to the longer length of treatment (3 to 5 years) necessary for psychoanalysis to cure phobic behavior. Although Lazarus (1971) reported that some patients showed a relapse 1 to 3 years after therapy, the renewed anxiety could be readily reversed with additional desensitization. The range of phobias successfully extinguished by desensitization is impressive: fears of heights, driving, snakes, dogs, insects, tests, water, flying, rejection by others, crowds, enclosed places, and injections are a few in a long list. In addition, desensitization apparently can be used with any behavior disorder initiated by anxiety. For instance, desensitization should help treat an alcoholic whose drinking occurs in response to anxiety. In general, research has demonstrated that systematic desensitization is a very effective way to successfully treat phobic behavior (Hersen & Rosqvist, 2005).

However, systematic desensitization therapy requires that patients be able to vividly imagine the fearful scene. Approximately 10 % of patients cannot imagine the phobic object sufficiently to experience anxiety (Masters et al., 1987); for these patients, another form of therapy is needed. Further, Rachman (1990) observed that therapy is more effective when a patient confronts a real, rather than imagined, phobic object. Imagined scenes are used in the initial phase of systematic desensitization in order to control the duration of exposure and prevent the association of the phobic objects with anxiety. You might be wondering

whether a patient can confront a phobic object but be able to control the duration of exposure to that object. Fortunately, modern technology appears to have made this possible. What is that technology? Perhaps you guessed: the use of a virtual reality environment.

Rothbaum, Hodges, Kooper, and Opdyke (1995) evaluated whether graded exposure to height-related stimuli in a virtual reality environment could effectively treat acrophobia. The researchers constructed a number of height-related stimuli, such as standing on a bridge, standing on a balcony, or riding in a glass elevator. The height of the stimuli varied: The bridge could be up to 80 meters above water, while the balcony or elevator could be as high as 49 floors.

Rothbaum et al. (1995) reported that a graded virtual reality exposure to height-related stimuli was an effective treatment for acrophobia. Following treatment, patients were able to stand on a real bridge or balcony or ride in a glass elevator. Similarly, Rothbaum, Hodges, Anderson, Price, and Smith (2002) reported that a virtual reality environment was as effective as a standard exposure treatment for fear of flying, while Garcia-Palacios, Hoffman, Carlin, Furness, and Botella (2002) found that a virtual reality environment can be used to treat a spider phobia. Virtual reality is a relatively new technology. As the technology improves, it seems highly likely that its use in systematic desensitization treatment will become more widespread.

Explorations for the Future

Desensitization is a well-established application of Pavlovian conditioning. Researchers are developing new applications based on current research; we will look at two of these new applications in this section.

An Intense Craving

In Chapter 3, we discovered that animals and people experience withdrawal following a drug exposure. The withdrawal from the drug can be intense and can act to motivate continued use of the drug. An opponent withdrawal state can be conditioned to the environmental cues surrounding drug administration, and exposure to these cues can produce withdrawal as a conditioned response. The **conditioned withdrawal response** produces a drug craving, which then motivates use of the drug; the greater the intensity of the withdrawal response, the greater the craving and the higher the likelihood of continued drug use.

Can an environmental stimulus produce withdrawal symptoms? Wikler and Pescor (1967) demonstrated that the conditioned withdrawal reaction can be elicited even after months of abstinence. They repeatedly injected dogs with morphine when the animals were in a distinctive cage. The addicted dogs were then allowed to overcome their unconditioned withdrawal reaction in their home cages and were not injected for several months. When placed in the distinctive cages again, these dogs showed a strong withdrawal reaction, including excessive shaking, hypothermia, loss of appetite, and increased emotionality.

Why is it so difficult for an addict to quit using drugs? Whenever an addict encounters the cues associated with a drug (e.g., the end of a meal for a smoker), a conditioned withdrawal will be elicited. The experience of this withdrawal may motivate the person to resume taking the drug. According to Solomon (1980), conditioned withdrawal reactions are what make eliminating addictions so difficult.

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Any substance abuse treatment needs to pay attention to conditioned withdrawal reactions. To ensure a permanent cure, an addict must not only stop “cold turkey” and withstand the pain of withdrawal, he or she must also extinguish the conditioned withdrawal reactions that all of the cues associated with the addictive behavior produce. Ignoring these conditioned withdrawal reactions increases the likelihood that addicts will eventually return to their addictive behavior. Consider the alcoholic who goes to a bar just to socialize. Even though this alcoholic may have abstained for weeks, the environment of the bar can produce a conditioned withdrawal reaction and motivate this person to resume drinking.

Can exposure to drug-related stimuli enhance an addict’s ability to avoid relapse? Charles O’Brien and his colleagues (Childress, Ehrman, McLellan, & O’Brien, 1986; Ehrman, Robbins, Childress, & O’Brien, 1992) have addressed this issue. Childress et al. (1986) repeatedly exposed cocaine addicts to the stimuli they associated with drug taking. Extinction experiences for these cocaine abusers involved watching videotapes of their “cook-up” procedure, listening to audiotapes of cocaine talk, and handling their drug paraphernalia. Childress et al. reported that their patients’ withdrawal responses and craving for drugs decreased as a result of exposure to drug-related cues. Further, the extinction treatment significantly reduced the resumption of drug use.

Other researchers (Higgins, Budney, & Bickel, 1994; Kasvikis, Bradley, Powell, Marks, & Gray, 1991) also have reported that exposure to drug-related stimuli reduced drug craving and consumption. However, the therapeutic success seems to be relatively short-lived; that is, drug craving returned with a subsequent reinstatement of drug use (O’Brien, Childress, Ehrman, & Robbins, 1998).

Why does drug craving and drug use return? Di Ciano and Everitt (2002) suggested that spontaneous recovery of drug craving occurs following extinction and results in the relapse of drug taking seen in treatment studies. To support this view, they trained rats to self-administer cocaine and then extinguished the cues present during cocaine consumption. After a 7-day extinction period, rats were tested after either 1 or 28 days. While the level of response to cocaine was significantly reduced 1 day after extinction, response significantly increased on the 28-day test. Di Ciano and Everitt assumed that spontaneous recovery of drug craving (CR) was responsible for the resumption of drug use on the 28-day test. The results of these studies indicate the important role the environment plays in motivating addictive behavior. They also suggest that for an extinction procedure to play a more effective role in the treatment of addictive disorders, the spontaneous recovery of drug craving needs to be eliminated, perhaps by repeated extinction trials over a longer period of time.

The Conditioning of an Immune System Response

Robert Ader and Nathan Cohen (Ader, 2001; Ader & Cohen, 1981, 1993) discovered that environmental events can suppress the functioning of the immune system. Interestingly, they made this discovery by accident. Following the pairing of saccharin-flavored water (CS) with cyclophosphamide (UCS), a drug which produces nausea, Ader and Cohen extinguished the aversion to the saccharin-flavored water. They reported that some of their animals died as a result of the presentation of the CS without the UCS.

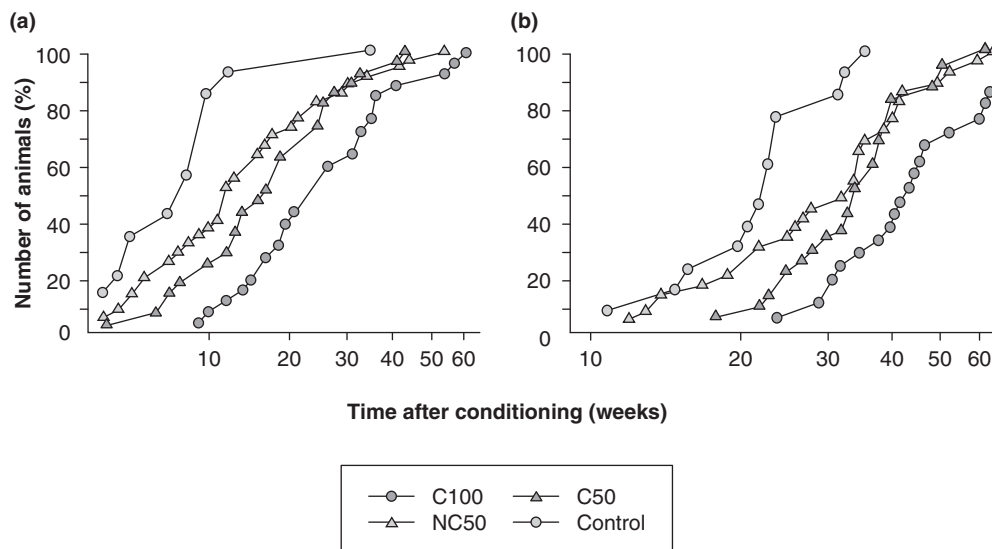
Why would presentation of saccharin-flavored water kill some of their animals? Ader and Cohen (1981) recognized that cyclophosphamide not only produces nausea but also suppresses the immune system. Perhaps the association of saccharin and cyclophosphamide resulted in the conditioning of immune system suppression as well as of nausea.

Ader and Cohen (1981) tested the idea that **conditioned immune system suppression** was responsible for the deaths of animals exposed to the saccharin-flavored water CS. These researchers injected animals with red blood cells from sheep. This alien substance normally activates the animals' immune systems and produces high levels of antibodies. Following injection of the red blood cells, some animals were presented with saccharin paired with cyclophosphamide. Other animals did not experience the saccharin-cyclophosphamide pairing. All animals then received several saccharin-alone extinction trials. Ader and Cohen reported that administering saccharin during extinction produced significantly fewer antibodies in those animals that had received saccharin paired with cyclophosphamide than in animals that had not experienced saccharin and cyclophosphamide. These results indicate that exposure to an environmental event (saccharin) associated with a drug (cyclophosphamide) produced immune system suppression as a CR. Other researchers (Blom, Tamarkin, Shiber & Nelson, 1995; Mei, Li, Deng, Sun, Ding, & Fan, 2000) have also reported that a flavor paired with cyclophosphamide becomes able to suppress immune system functioning.

Cyclophosphamide is not the only immunopharmacologic agent that has been used to produce conditioned immune system suppression. Coussons, Dykstra, and Lysle (1992) paired a distinctive environmental stimulus with morphine, a drug that suppresses several nonspecific immune responses. Following conditioning, exposure to the environmental stimulus produced a conditioned immune system suppression. Other immunopharmacologic agents that can produce conditioned immunosuppression include corticosteroids (Kusnecov, Husband, & King, 1990) and antilymphocyte serum (Kusnecov, Sivyer, King, Husband, Cripps, & Clancy, 1983). Electric shock also can cause immune system suppression, and the stimuli associated with a shock stressor will become able to produce conditioned immune system suppression (Zalcman, Irwin, & Anisman, 1991).

The conditioning of immune system functions could be used to treat diseases of the immune system. The drug cyclophosphamide is used to suppress the immune system as part of the treatment for lupus, a disorder in which the immune system causes the body to attack itself. Yet cyclophosphamide has some seriously debilitating side effects. Perhaps the dose of cyclophosphamide could be reduced and its use supplemented with a psychological treatment to suppress the immune system. Ader and Cohen (1982) provided support for this idea by showing that the conditioning of immune system suppression delayed the development of systemic lupus erythematosus in New Zealand mice. In this study, the experimental group of female mice received one trial of saccharin-cyclophosphamide pairing each week. Ader and Cohen found that these experimental animals showed a slower rate of lupus progression and a lower mortality than both animals that did not experience trials of saccharin paired with cyclophosphamide and animals that experienced trials of saccharin without cyclophosphamide. Some retardation of illness was seen in animals that experienced an equal number of saccharin-cyclophosphamide and saccharin-saline pairings. Figure 5.9 presents the results of this study.

FIGURE 5.9 The conditioned suppression of the immune system slows the progression of the autoimmune disease lupus erythematosus in New England mice. The pairing of a saccharin solution with an injection of cyclophosphamide each week delayed the onset of disease (proteinuria, A) and lowered mortality levels (B) in most animals in Group C100. By contrast, animals that received unpaired saccharin and cyclophosphamide exposures (Group NC50) did not differ in terms of either the onset of the proteinuria disease or mortality rate compared to mice that never received cyclophosphamide (Control Group). However, animals that received an equal number of saccharin-cyclophosphamide and saccharin-saline exposures (Group C50) showed some delayed disease onset and lowered mortality



Can Pavlovian conditioning be used in the treatment of lupus? Olness and Ader (1992) described their successful application of Pavlovian conditioning to the treatment of severe lupus in an 11-year-old girl. Their treatment consisted of six pairings of a taste (cod liver oil) and smell (rose perfume) with cyclophosphamide over a 12-month period. The pairings were given every other month. (The usual treatment would have consisted of 12 cyclophosphamide treatments.) The girl received a taste-only experience between each cyclophosphamide treatment. Olness and Ader found a significant reduction in the symptoms of lupus in the girl during the 12-month treatment period. Also, she continued to do well when evaluated 5 years after the conditioning treatment ended. While the results of a single case study must be viewed with caution, this successful application of Pavlovian conditioning suggests an important breakthrough in the treatment of this immune system disorder.

In several diseases in which the immune system is suppressed, such as acquired immune deficiency syndrome (AIDS), psychologists have attempted to condition increased immune system functioning. Several studies (Demissie, Ghanta, Hiramoto, & Hiramoto, 2000; Gee, Thiele, & Johnson, 1994) have reported **conditioned immune system enhancement** in mice. In a study involving human subjects, Buske-Kirschbaum, Kirschbaum, Stierle, Jabaj, and Hellhammer (1994) paired a sherbet flavor with injections of adrenaline. One unconditioned response to adrenaline is increased activity of natural killer (NK) cells of the immune system. Following several sherbet-adrenaline pairings, the sherbet elicited an enhanced immune system response. While it seems clear that immune system enhancement can be conditioned, additional research is needed to determine its applicability to the treatment of AIDS and other diseases involving immune system suppression.

BEFORE YOU GO ON

- How might systematic desensitization be used to overcome Juliette's fear of darkness (see Chapter 4 opening vignette)?
- What might be the effect of Juliette's fear on her immune system?

SECTION REVIEW

- Systematic desensitization is a graduated counterconditioning procedure to eliminate phobias.
- The patient first constructs a hierarchy of feared stimuli; relaxation is then paired with the feared stimuli.
- The counterconditioning process begins with imagining the least feared stimulus for a brief time, with the time increased until the patient can imagine it without any discomfort, and continues until the patient can imagine the most aversive stimulus without becoming anxious.
- The success of desensitization is considerable; the elimination of fear of heights, driving, tests, flying, and enclosed places are a few examples of its successful application.
- As part of a treatment program for addiction, exposure to the stimuli associated with drug use can extinguish the conditioned craving and reduce the likelihood of continued use.
- Conditioned immune system suppression represents a potential application of Pavlovian conditioning in the treatment of lupus and other diseases of the immune system, while conditioned immune system enhancement may represent an application of Pavlovian conditioning in the treatment of AIDS.

CRITICAL THINKING QUESTIONS

1. Yancy initially experienced intense euphoria after injecting heroin. His response to heroin is now much less intense. Using Siegel's research, give an explanation for Yancy's current reaction to heroin. What would happen if Yancy injected heroin while he was in a new place?
2. Diane becomes ill after drinking several beers, yet she does not develop an aversion to it. Describe the process(es) responsible for these preexposure effects.

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3. Greg has an intense desire to smoke cigarettes. His nicotine craving occurs after a meal, a class, or a movie, as well as at other times. Describe the process responsible for Greg's craving. How might Greg eliminate his craving? What problems might Greg encounter? How can he avoid these problems?

KEY TERMS

affective extension of SOP theory (AESOP)	hypoalgesia
analgesia	learned irrelevance
backward blocking	Mackintosh's attentional view
comparator theory	overshadowing
conditioned emotional reaction	phobia
conditioned immune system enhancement	potentiation
conditioned immune system suppression	reciprocal inhibition
conditioned withdrawal response	Rescorla-Wagner associative model
context blocking	retrospective processing
CS preexposure effect	sometimes-opponent-process (SOP) theory
cue-controlled relaxation	spatial-temporal hierarchy
cue deflation effect	stimulus-substitution theory
drug tolerance	systematic desensitization
hyperalgesia	thematic hierarchy
	UCS preexposure effect
	within-compound association